A STUDY ON CARDIO EMBOLIC STROKE – CLINICAL PROFILE, ARTERIAL TERRITORY AND TYPE OF CARDIAC LESION

DISSERTATION SUBMITTED FOR M.D. DEGREE EXAMINATION OF

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CERTIFICATE

This is to certify that this dissertation entitled **'Cardio embolic stroke – study of clinical profile, Arterial territory and Type of cardiac lesion'** submitted by **Dr.P.Rajavel Murugan** to the faculty of Medicine, The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD Degree Branch I (General Medicine), is a bonafide research work carried out by him under our direct supervision and guidance.

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CONTENTS

INTRODUCTION

Embolic stroke is recognized increasingly as an important cause of stroke. This is the commonest cause of stroke. The characteristic feature is the abrupt onset of a focal neurologic deficit. In most cases of cerebral embolism, the embolic material consists of a fragment that has broken away from a thrombus within the heart. Somewhat less frequently the source is intra-arterial, from the distal end of a thrombus within the lumen of an occluded or severely stenotic carotid or vertebral artery or the distal end of a carotid dissection, or possibly from an atheromatous plaque that has ulcerated into the lumen of the carotid sinus. Single or sequential emboli may also arise from large atheromatous plaques in the ascending aorta. Thrombotic or infected material (endocarditis) that adhere to the aortic or mitral heart valves and break away are also well appreciated sources of embolism. [(1)]

Cardioembolic stroke accounts for approximately 15% of all strokes and is thought to be one of the more preventable types of strokes[2]. A study published in 1989[(3)] reported a cardioembolic mechanism in 23.5% of 540 consecutive stroke patients, whereas the

German Stroke Data Bank,[4] published in 2001, reported a cardioembolic mechanism in 25.6% of patients. [2,6]

This cardioembolic stroke is largely preventable, making measures of primary prevention valuable. Once stroke secondary to cardiac embolism has occurred, the likelihood of recurrence is high; thus secondary prevention is also equally important.

The cardiac lesions causing the stroke have great importance in morbidity and mortality of the illness. 75 percent of cardiogenic emboli lodge in the brain. However, the designation of an ischemic stroke as cardioembolic is usually presumptive based on the associated supportive factors at hand.[3] For example, the presence of atrial fibrillation in an older stroke patient makes cardioembolism the most likely mechanism until proved otherwise. Coexistent significant valvular heart disease would make the mechanism of cardioembolism even more likely. Conversely, a relatively normal echocardiogram and the presence of high-grade carotid stenosis ipsilateral to the infarct would make the mechanism of cardioembolism more questionable.

Significant cardiac lesion in the form of Rheumatic vascular heart disease or ischemic heart disease which predispose to formation of thrombus in cardiac chambers which inturn lead to recurrent embolic stroke. As per various studies embolic stroke is commonly caused by cardiac lesion which fact implies that this is preventable if cardiac lesion is promptly identified and cured. Atrial fibrillation either rheumatic or non rheumatic predisposes to embolic stroke as per text book and studies. So our study focuses on significance of cardiac lesion in etiology of embolic stroke.

AIM OF THE STUDY

1. To study the significance of the time of onset of cardioembolic stroke
2. To know the significance of activity at the time of onset
3. To identify the arteries involved in cardioembolic stroke
4. To study the type of cardiac lesion that commonly cause the stroke.
5. To correlate clinical findings with CT findings
6. To correlate the clinical types with specific cardiac lesion
7. To study the outcome in cardioembolic stroke

REVIEW OF LITERATURE

STROKE

Definition:

Stroke or CVA is a rapidly developing clinical symptoms and signs of focal and at times global loss of cerebral function with symptoms lasting for more than 48 hrs and leading to death with no apparent cause other than that of vasacular origin.

Infarction: (80%)

Large vessel

Small vessel

Cardiac source

Hematological disorder

Vasculopathy

Haemorrhage:

Hypertension/ vascular malformation / - 10%

bleeding disorder, anticoagulation , SAH - 5%

Other causes - 5%

Embolic stroke

Aetiology :

The commonest identifiable cause is chronic or recent atrial fibrillation, the source of the embolus being a mural thrombus within the atrial appendage.[5]

Patients with chronic atrial fibrillation are about six times more liable to stroke than an age matched population with normal cardiac rhythm.

Embolism may also occur during paroxysmal atrial fibrillation or flutter. Mural thrombus deposited on the damaged endocardium overlying a myocardial infarct in the left ventricle, particularly if there is an aneurysmal sac, is an important source of cerebral emboli, as is a thrombus associated with severe mitral stenosis without atrial fibrillation.

Emboli tend to occur in the first few weeks after an acute myocardial infarction. Cardiac catheterization or surgery, especially valvuloplasty, may disseminate fragments from a thrombus or a

calcified valve. Mitral and aortic valve prostheses are additional important sources of embolism.

1. Atrial fibrillation and other arrhythmias

2. Myocardial infarction with mural thrombu

3. Acute and subacute bacterial endocardit\is

4. Heart disease without arrhythmia or mural thrombus (mitral stenosis, myocarditis, etc.)

5. Complications of cardiac surgery

6. Valve prostheses

7. Nonbacterial thrombotic (marantic) endocardial vegetations

8. Prolapsed mitral valve

9. Paradoxical embolism with congenital heart disease (e.g.,patent foramen ovale)

10. Myxoma

11.Nonischemic dilating cardiomyopathies

12.Amyloid deposition

13.Hypereosinophilia

14.Sarcoidosis

15.Alcoholic sequelae

16.Catecholamine induced

17.Chagas disease

18.Doxorubicin

19.Crack cocaine use

20.Oxalosis

21.Peripartum effect

22.Cardiac tumors

- Atrial myxoma
- Cardiac sarcoma
- Endocardial fibroelastoma
- Metastatic disease

PATHOPHYSIOLOGY:

No single mechanism is responsible for the development of cardiac emboli. The underlying cardiac disease determines the pathophysiology. Emboli secondary to chamber abnormalities (eg, atrial fibrillation, acute myocardial infarction) are induced mainly by stasis, while those secondary to valve involvement are the result of endothelial abnormalities with attachment of material (eg, platelets, bacteria) to their free borders. The nature of the embolus differs

depending on the source (eg, calcified particles from calcific valves, neoplastic cells from myxomas).

Emboli from the heart are distributed evenly throughout the body according to cardiac output, but more than 80% of symptomatic or clinically recognized emboli involve the brain. Of emboli to the brain, approximately 80% involve the anterior circulation, proportional to the distribution of cerebral blood flow.[3,5.11,13]

Once emboli have reached the cerebral circulation, they obstruct brain-supplying arteries, causing ischemia to the neurons and to the blood vessels within the area of ischemia. In contrast to thrombi, emboli are attached loosely to the vascular walls and thus commonly migrate distally.

When this occurs, reperfusion of the damaged capillaries and arterioles allows blood to leak into the surrounding infarcted tissue. This explains in part the more frequent association of hemorrhagic infarctions with embolism than with other causes of ischemic stroke. In the great majority of patients with hemorrhagic infarcts, the hemorrhagic transformation does not cause clinical worsening, since the bleeding involves necrotic tissue.[2,6]

In short, cardioembolic stroke is not one disease with a single natural history. Many different types of cardiac disorders lead to cardioembolic stroke, each with unique clinical features, risks of initial and recurrent stroke, and optimal therapy.

Clinical features :

- Decreased level of consciousness at onset of stroke
- Sudden onset of symptoms and signs that are maximal at onset
- Rapid recovery from major hemispheric deficits (”spectacular shrinking deficit”) due to reperfusion of brain with early lysis of the embolus
- Onset of symptoms after a Valsalva maneuver (patent foramen ovale)
- Symptoms reflecting involvement of different vascular territories of the brain
- Cortical deficit (eg, aphasia-visual field defect) involving mainly the middle cerebral arteries and the posterior cerebral arteries and their branches

- Cardiogenic emboli (especially from valvular sources) do not often affect the deep penetrating arteries or present as a lacunar syndrome.[1,2,6]

Physical:

- Evidence of cardiac dysrhythmias (eg, atrial fibrillation, sick sinus syndrome)
- Presence of cardiac murmurs (eg, mitral stenosis, calcific aortic stenosis)
- Signs of congestive heart failure (eg, after acute myocardial infarction, nonischemic cardiomyopathies)
- Concomitant diseases (eg, systemic lupus erythematosus and Libman-Sachs endocarditis, neoplasia, marantic endocarditis)

Diagnosis is based on the triad of

(1) identification of a potential cardioembolic source,

(2) absence of other likely causes of stroke,

(3) support of specific clinical features described above.

Risk factors :

High risk : Atrial fibrillation

Mechanical valve

Anterior wall MI

Mitral stenosis

Thrombus

Endocarditis

Medium risk:

LV aneurysm

Bioprosthetic valve

CCF

Cardiomyopathy

Low risk :

PFO

Atrial septal aneurysm

Atrial Fibrillation

AF is the most common sustained arrhythmia. It is marked by disorganized, rapid, and irregular atrial activation. The ventricular response to the rapid atrial activation is also irregular. In the untreated patient, the ventricular rate also tends to be rapid and is entirely dependent on the conduction properties of the AV junction. The rate can be >200 beats/min. In other patients, because of heightened vagal tone or intrinsic AV nodal conduction properties, the ventricular response is <100 beats/min and occasionally even profoundly slow. The impulse appears to originate predominantly from the atrialized musculature that enters the pulmonary veins. [1,3,15]

Mechanism :

focal abnormal automaticity

triggered firing that is somewhat modulated by autonomic influences.

Incidence :

AF increases with age such that >5% of the adult population over 70 will experience the arrhythmia. As many patients are asymptomatic with AF, it is anticipated that the overall incidence,

particularly that noted in the elderly, may be more than double previously reported rates. [1]

Causes :

Rheumatic Valvular heart disease – mitral especially

Infective endocarditis

Pericarditis

Cardiomyopathy

acute hyperthyroidism,

acute vagotonic episode,

acute alcohol intoxication.

recovery phase of major vascular, abdominal, and thoracic surgery.

AF may also be triggered by other supraventricular tachycardias such as AV nodal reentrant tachycardia (AVNRT).

Clinical importance :

1. The loss of atrial contractility,
2. The inappropriate fast ventricular response,

3. The loss of atrial appendage contractility and emptying leading to the risk of clot formation and subsequent thromboembolic event

Features :

- ❖ Asymptomatic and no apparent hemodynamic consequences to the development of AF.
- ❖ Minor palpitations or sense irregularity of their pulse.
- ❖ Severe palpitations.
- ❖ Hypotension, pulmonary congestion, and anginal symptoms may be severe in some..
- ❖ In LV diastolic dysfunction such as hypertension, hypertrophic cardiomyopathy, or obstructive aortic valvular disease, symptoms may be even more dramatic.
- ❖ Exercise intolerance and easy fatigability are the hallmarks of poor rate control with exertion.
- ❖ Occasionally, the only manifestation of AF is severe dizziness or syncope.
- ❖ The ECG in AF is characterized by the lack of organized atrial activity and the irregularly irregular ventricular response.

- Lead V_1 may frequently show the appearance of organized atrial activity that mimics AFL because the lateral right atrium activated by a more uniform activation wavefront that originates over the roof of the right atrium.
- ECG assessment of the PP interval (<200 ms) and the chaotic P wave morphology in the remaining ECG leads will confirm the presence of AF.
- Evaluation of the patient with AF should include a search for reversible causes of the arrhythmia, such as hyperthyroidism or anemia.[1]
- An echocardiogram should be performed to determine if there is structural heart disease.
- Persistent or labile hypertension should be identified and treated, and heart failure treatment should be optimized.

Risk Factors for Stroke in Atrial Fibrillation [1]

History of stroke or transient ischemic attack Mitral stenosis Hypertension Diabetes mellitus	Age > 75 years Congestive heart failure Left ventricular dysfunction Marked left atrial enlargement (>5.0 cm) Spontaneous echo contrast

Treatment:

Factors to be considered

- Clinical situation in which the arrhythmia is encountered
- The chronicity of the af
- The status of the patient's level of anticoagulation
- Risk factors for stroke
- The patient's symptoms
- The hemodynamic impact of the AF, and the ventricular rate.

Acute Rate Control

In the absence of hemodynamic compromise ,

The initial goals of therapy are

(1) to establish control of the ventricular rate

(2) to address anticoagulation status

Begin IV heparin treatment if the duration of AF is >12 h and risk factors for stroke with AF are present

Ventricular rate control with beta blockers and/or calcium channel blocking agents, verapamil or diltiazem.

Digoxin may add to the rate-controlling benefit of the other agents (uncommonly used as a stand-alone agent)

Anticoagulation

In patients who have known risk factors for stroke as mentioned in the table.

Chronic anticoagulation with warfarin targeting an INR between 2.0 and 3.0 is recommended in patients with persistent or frequent AF and risk factors.

If patients have not been adequately anticoagulated and the AF is more than 24–48 h in duration, a transesophageal echocardiogram (TEE) can be performed to exclude the presence of a left atrial thrombus that might dislodge with the attempted restoration of sinus rhythm using either nonpharmacologic or pharmacologic therapy.[1,10]

Anticoagulation must be instituted coincident with the TEE and maintained for at least 1 month following restoration of sinus rhythm.

Heparin is maintained routinely until the INR is 1.8 with the administration of warfarin after the TEE.

For patients who do not warrant early cardioversion of AF, anticoagulation should be maintained for at least 3 weeks with the INR confirmed to be >1.8 on at least two separate occasions prior to attempts at cardioversion.[1,15,26]

Termination of AF:

Acutely may be warranted based on clinical parameters and/or hemodynamic status.

Conversion rates using a 200-J biphasic shock delivered synchronously with the QRS complex typically are >90%.

Pharmacologic therapy to terminate AF is less reliable.

Oral and/or IV administration of amiodarone or procainamide have only modest success.

The IV administration of ibutilide appears to be more effective and may be used in selected patients to facilitate termination with direct current (DC) cardioversion .

Arterial territory :

The neurologic picture will depend on the artery involved and the site of obstruction.

A large embolus may plug the distal internal carotid artery or the stem of the middle cerebral artery, producing the full-blown syndromes that follow occlusion of these arteries. More often the embolus passes into one of the branches of the middle cerebral artery, producing a strikingly focal disorder such as a motor speech disorder, a hemiplegia, or a receptive aphasia.(1,2,),

Anterior and Posterior cerebral arteries are affected at a lower frequency than the middle cerebral because of their lower rates of blood flow. [5,89,20]

The picture of total occlusion of the stem is one of contralateral hemiplegia (face, arm, and leg), hemianesthesia, and homonymous hemianopia (due to infarction of the lateral geniculate body), with deviation of the head and eyes toward the side of the lesion; in addition, there is a variable but usually global aphasia with left hemispheric lesions and anosognosia and amorphosynthesis with a right-sided ones ACA involvement may cause crural monoplegia, bladder or bowel dysfunction. Mental changes, apraxia or impairment in executive function may also be features.[7,8,23]

PCA involvement usually causes poor vision and homonymous hemianopsia. Hemiplegia or hemianaesthesia may be the rare features. Impairment of memory, alexia and metamorphopsia may also be reported in some cases.[7,8]

MCA territory:

Major infarction in the territory of the superior division causes a dense sensorimotor deficit in the contralateral face, arm, and, to a

lesser extent, leg as well as ipsilateral deviation of the head and eyes; i.e., it mimics the syndrome of stem occlusion except that the foot is spared and the leg is less involved than the arm and face ("brachiofacial paralysis").

Occlusion of the inferior division of the middle cerebral artery is slightly less frequent than occlusion of the superior one, but again is nearly always due to embolism. The usual result in left-sided lesions is a Wernicke aphasia, which generally remains static for days or weeks, or a month or two, after which some improvement can be expected With either right-or left-hemispheric lesions, there is usually a superior quadrantanopia or homonymous hemianopia and, with right-sided ones, a left visual neglect and other signs of amorphosynthesis.

ACA territory:

Occlusion of the stem of the anterior cerebral ry, proximal to its connection with the anterior communicating artery (the A1 segment, in neuroradiologic parlance), is usually well tolerated, since adequate collateral flow will come from the artery of the opposite side. Maximal disturbance occurs when both arteries arise from one anterior cerebral stem, in which case occlusion of the stem will cause

infarction of the anterior and medial parts of both cerebral hemispheres and result in paraplegia, incontinence, abulia and motor aphasic symptoms, and frontal lobe personality changes.

Occlusion of the anterior cerebral arteries is usually embolic occlusion of one anterior cerebral artery distal to the anterior communicating artery (A2 segment) results in a sensorimotor deficit of the opposite foot and leg and,to a lesser degree, of the shoulder and arm, with sparing of the hand and face. The motor disorder is more pronounced in the foot and leg than in the hip and thigh. Sensory loss, when it occurs, is mainly of the discriminative modalities and is mild or absent in some cases. The head and eyes may deviate to the side of the lesion.

Urinary incontinence and a contralateral grasp reflex and paratonic rigidity (gegenhalten) may be evident.

The configuration and branches of the proximal segment of the **posterior cerebral artery**.

Thalamic syndrome: sensory loss (all modalities), spontaneous pain and dysesthesias, choreoathetosis, intention tremor, spasms of hand, mild hemiparesis.

Thalamoperforate syndrome: (1) superior, crossed cerebellar ataxia; (2) inferior, crossed cerebellar ataxia with ipsilateral third nerve palsy (Claude syndrome).

Weber syndrome—third nerve palsy and contralateral hemiplegia Contralateral hemiplegia.

Paralysis or paresis of vertical eye movement, skew deviation, sluggish pupillary responses to light, slight miosis and ptosis (retraction nystagmus and "tucked-in" eyelids may be associated).

Contralateral ataxic or postural tremor.

Peripheral territory - Homonymous hemianopia Bilateral homonymous hemianopia, cortical blindness, unawareness or denial of blindness; achromatopsia, failure to see toand- fro movements, inability to perceive objects not centrally located, apraxia of ocular movements, inability to count or enumerate objects.

Dyslexia without agraphia, color anomia, Memory defect Topographic disorientation and prosopagnosia Simultagnosia Unformed visual hallucinations, metamorphopsia, teleopsia, illusory visual spread, palinopsia, distortion of outlines, photophobia outcome.

Most patients survive the initial insult, and in many the neurologic deficit may recede relatively rapidly, as indicated above.

It is important to repeat that an embolus may produce a severe neurologic deficit that is only temporary; symptoms disappear as the embolus fragments. In other words, embolism is a common cause of a single evanescent stroke that may reasonably be called a prolonged TIA. The eventual prognosis is determined by the occurrence of further emboli and the gravity of the underlying illness—cardiac failure, myocardial infarction, bacterial endocarditis, malignancy, and so on.

In a small number of cases, the first episode of cerebral embolism will be followed by another, frequently with severe consequences if the second stroke affects the opposite hemisphere. There is no certain way of predicting when the second embolus will strike. However, the occurence of this second event, once thought to

be as high as 20 percent, has been revised downward, to perhaps 2 percent, based on several large trials that were designed to test the effects of anticoagulation (see the review of Swanson). This has a bearing on the choice of early treatment after embolic stroke.

MATERIALS AND METHODS

The study of cardio embolic stroke was carried out in department of Medicine , Tirunelveli Medical College. TVMCH is a tertiary care centre and a referral centre.

Settings :

Medical Wards

Study Design:

Single centre Observational Prospective hospital based study

Period of study:

December 2007 to April 2009

Materials :

All stroke patients admitted in the above period

Stroke patients who satisfied the set criteria

Inclusion criteria;

1. Stroke as defined by WHO

2. All patients with CT proven case of ischemic stroke

3. All stroke patients with ECG or Echo evidence of cardiac lesion

4. Do not satisfy the exclusion criteria

Exclusion criteria:

1. Patients with TIA
2. Patients with haemorrhagic stroke
3. CT negative stroke
4. Patients with nomal heart as evidenced by Clinical examination, ECG and Echo
5. Patients with major renal, hepatic and cancerous disease.
6. Stroke patients with lab evidence of SLE

Methodology :

After obtaining verbal consent from either patient's relatives all patients were evaluated by complete medical history, full CNS examination, CVS examination and imaging studies

History :

Clinical history was recorded from the patient or relatives. Special emphasis was given to presenting complaint, mode of onset, presence or absence of seizures, headache, vomiting. Past history of IHD, Rheumatic heart disease were carefully sought.

General Examination:

Apart from routine, markers of atherosclerosis, CHD,Marfan's were noted

CNS examination:

Each patient was assessed according to fixed protocol. First evaluation was done 24hrs after admission. A detailed clinical profile such as aphasia, cranial nerve palsies, limb weakness, sensory impairment, cerebellar dysfunction, were elicited by standard comprehensive bedsisde CNS examination.

Detailed CVS examination to find out congenital heart disease, valvular heart disease were carried out.

Investigations :

Apart from routine baseline investigations patients were assessed for cardiac lesions with Xray chest, ECG.

Cardiologist opinion was obtained.

Echo was done to assess severity of lesion,rule out mural thrombus and vegetations.

CT remains the traditional initial neuroimaging procedure for patients who present with manifestations of acute stroke.

CT brain Plain and contrast was done to locate site of infarct and identify artery involved

Follow up:

Re examination was done on 5th day to assess clinical status of the patient and condition reviewed. Patients motor system was assessed with Barthell score to look for any improvement in power. Proper nursing care and physiotherapy were explained to the relatives, care givers and when possible to the patients. Then 3rd evaluation was done at 4th week of followup. Improvement was assessed by determining the functional status.

Doubts and apprehensions of the relatives, care givers and patients were addressed and cleared. Importance of continuing cardiac drugs, anti coagulants, nursing care and physiotherapy were reemphasized.

BARTHEL INDEX OF ACTIVITY OF DAILY LIVING

1.	Feeding	10= Independent 5 = Needs help (i.e) forcutting 0 = Inferior performance
2.	Bathing	5 = Performs without assistance 0 = Inferior performance
3.	Personal toilet	5 = Washes face, combs hair, brushes teeth
4.	Dressing	10 = Independent 5 = Needs help 0 = Inferior performance
5.	Bowel control	10 = No accidents 5 = Occasional accidents 0 = Inferior performance
6.	Bladder control	10 = No accidents 5 = Occasional accidents 0 = Inferior performance
7.	Toilet transfer	10 = Independent with toilet or bed pan 5 = Needs help for balance 0 = Inferior performance

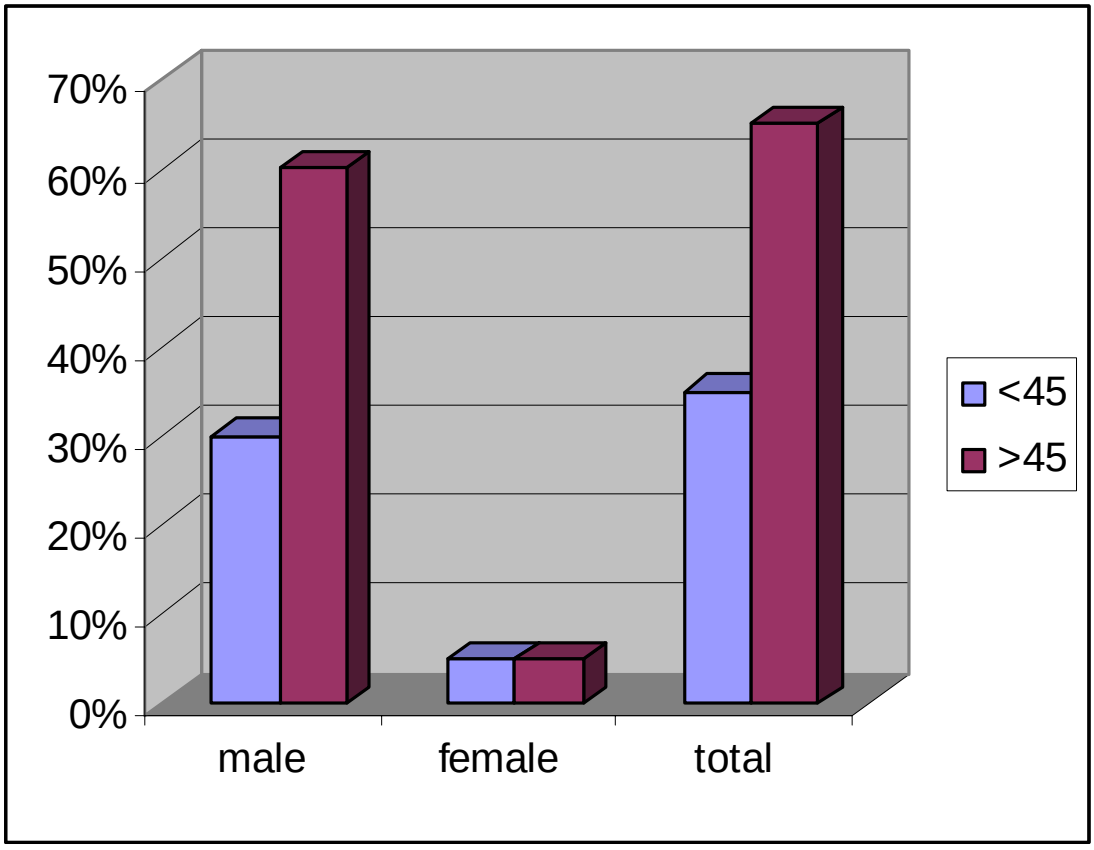
8.	Chair / bed transfers	15 = Independent 10 = Minimum assistance 5 = Able to sit, Needs assistance to transfer 0 = Inferior performance
9.	Ambulation	15 = Independent for 50 yards 10 = With help for 50 yards 5 = Independent with wheelchair for 50 yards, only if unable to walk 0 = Inferior performance
10.	Stair climbing	10 = Independent 5 = Needs help or Supervision 0 = Inferior performance
	Maximum disability	= 0
	Minimum disability	= 100

OBSERVATION AND RESULTS

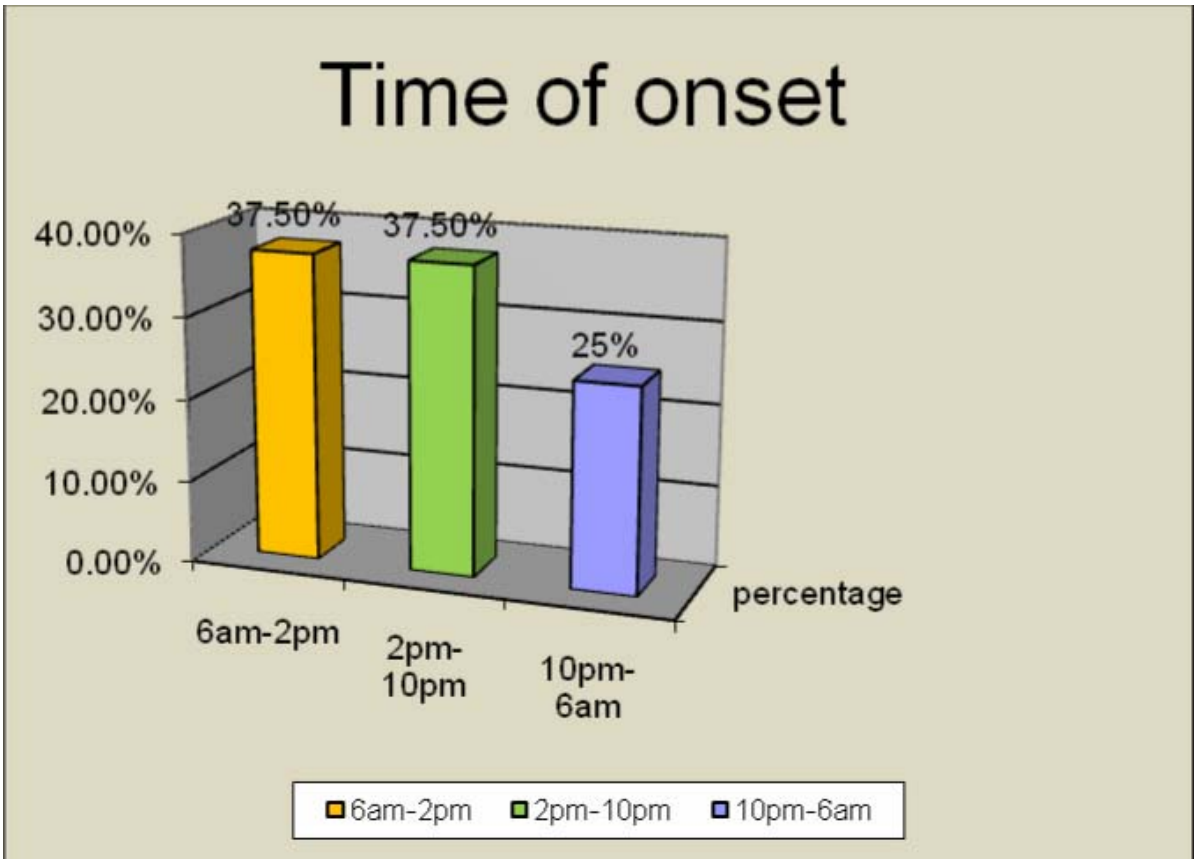
40 patients who satisfied all the above criteria were included in the study. 36 males and 4 females were selected according to the criterias. Following observations were made out of 40 patients at the time of admission.

All the patients were admitted with hemiparesis or hemiplegia of sudden onset . The weakness was maximum at its onset in all patients as in any other case of embolic stroke. Out of 40 cases 22 patients were known to have rheumatic valvular hear disease who were fully evaluated in the past. Eventhough typical rheumatic history was absent in some of the patients, more than 20 cases were proved to be rheumatic according to their old records. The earliest age reported was 14 atwhich patient had symptoms of rheumatic fever. All the patients were subjected to cardiac evaluation and CT brain. CT findings of all the patients proved ischemic infarct in any of the arterial territory.

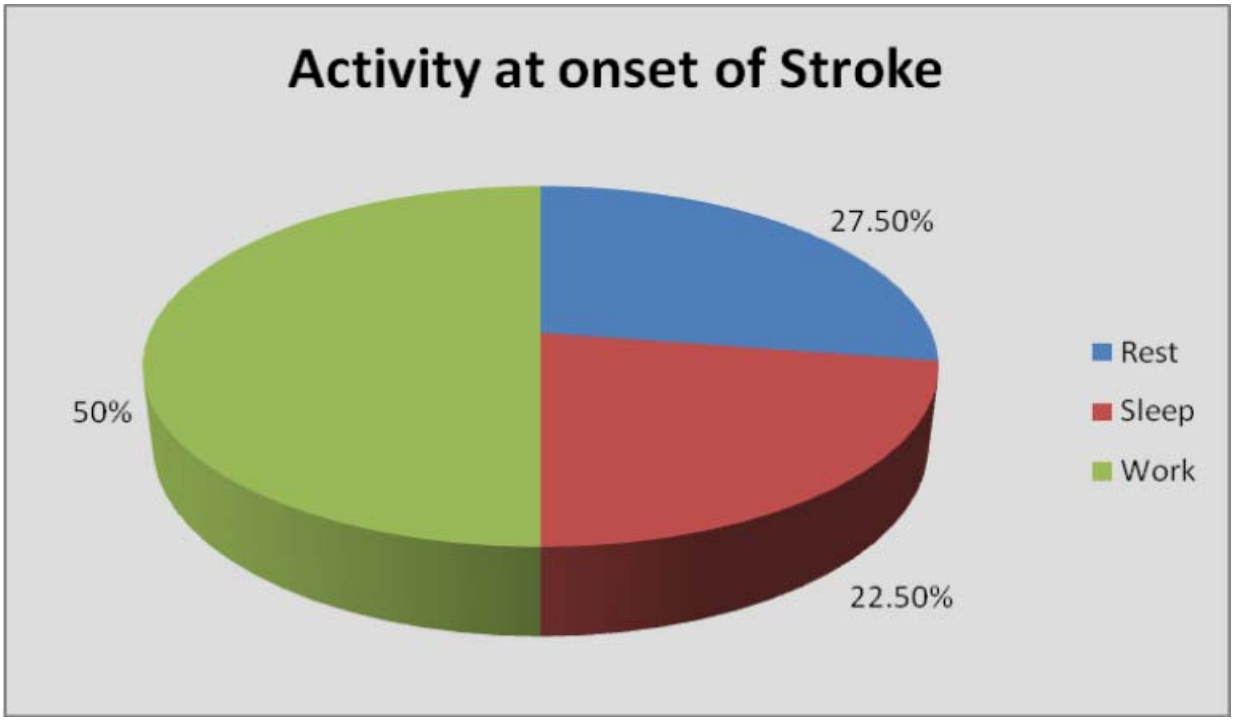
Sex distribution

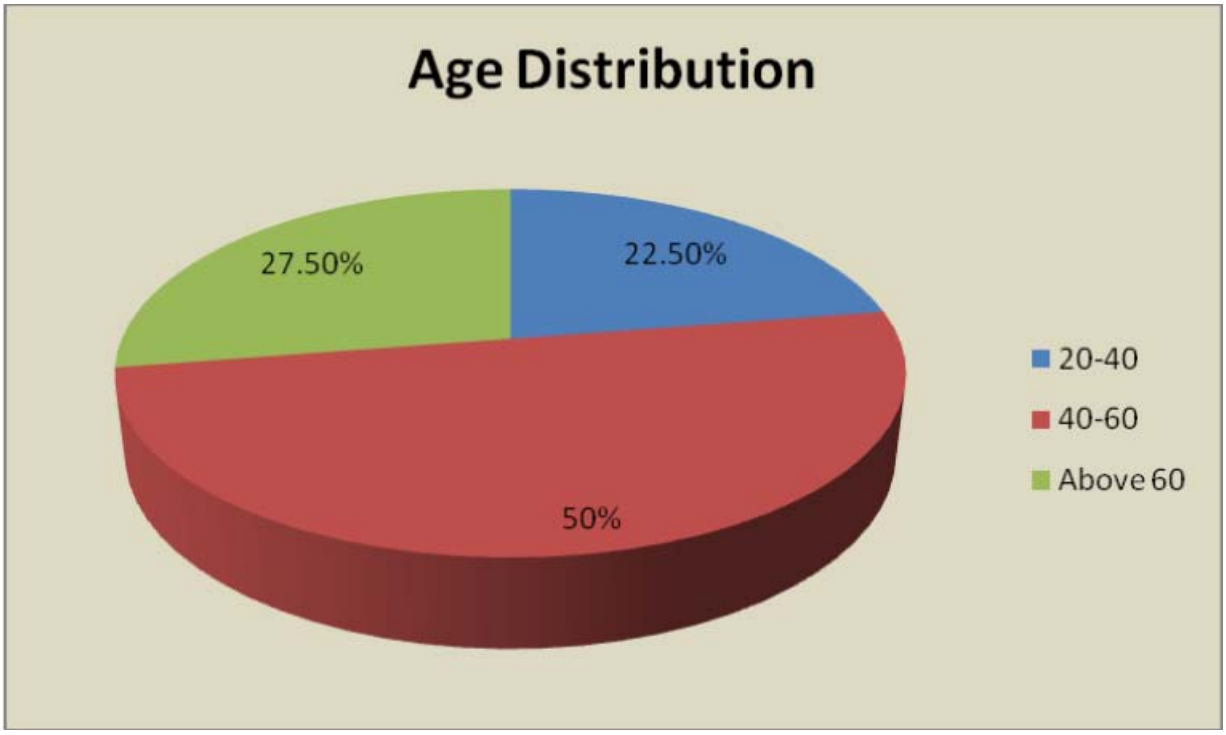


Time of onset	No of patients	Percentage
6.00am to 2.00pm	15	37.5%
2.00pm to 10.00pm	15	37.5%
10.00pm to 6.00am	10	30.0%

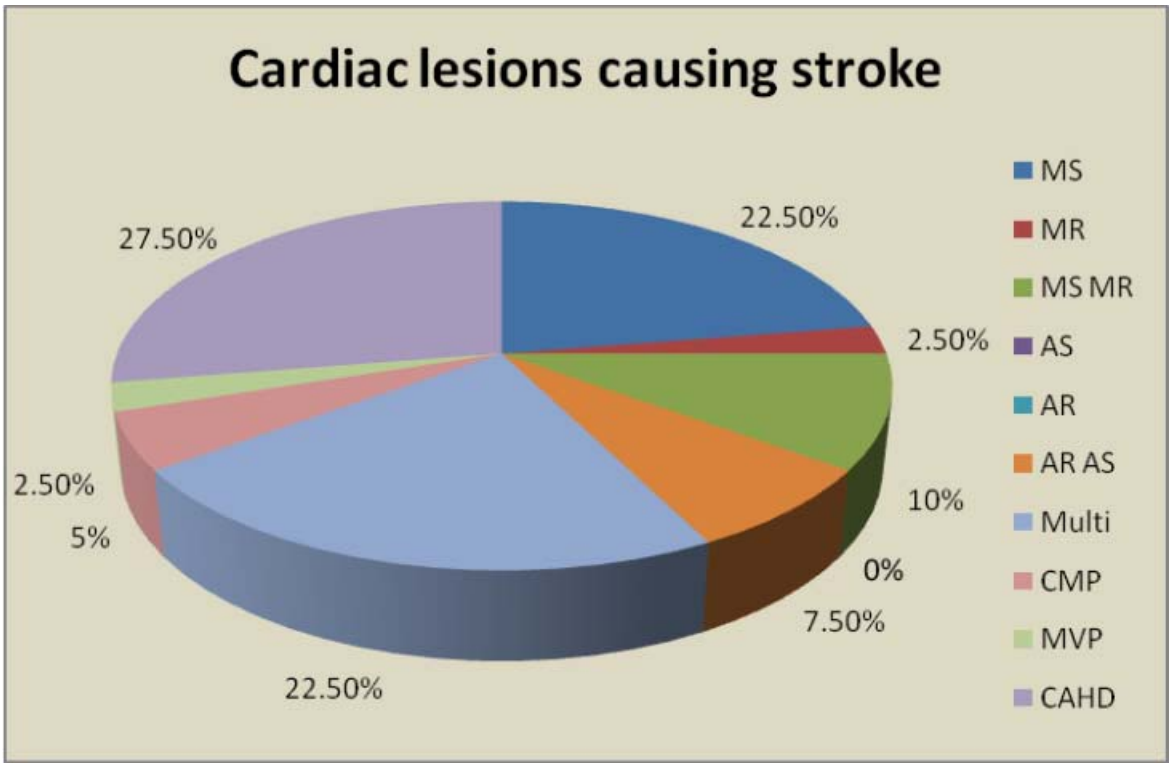


Reset	Sleep	Work
11	9	20
27.5%	22.5%	50%

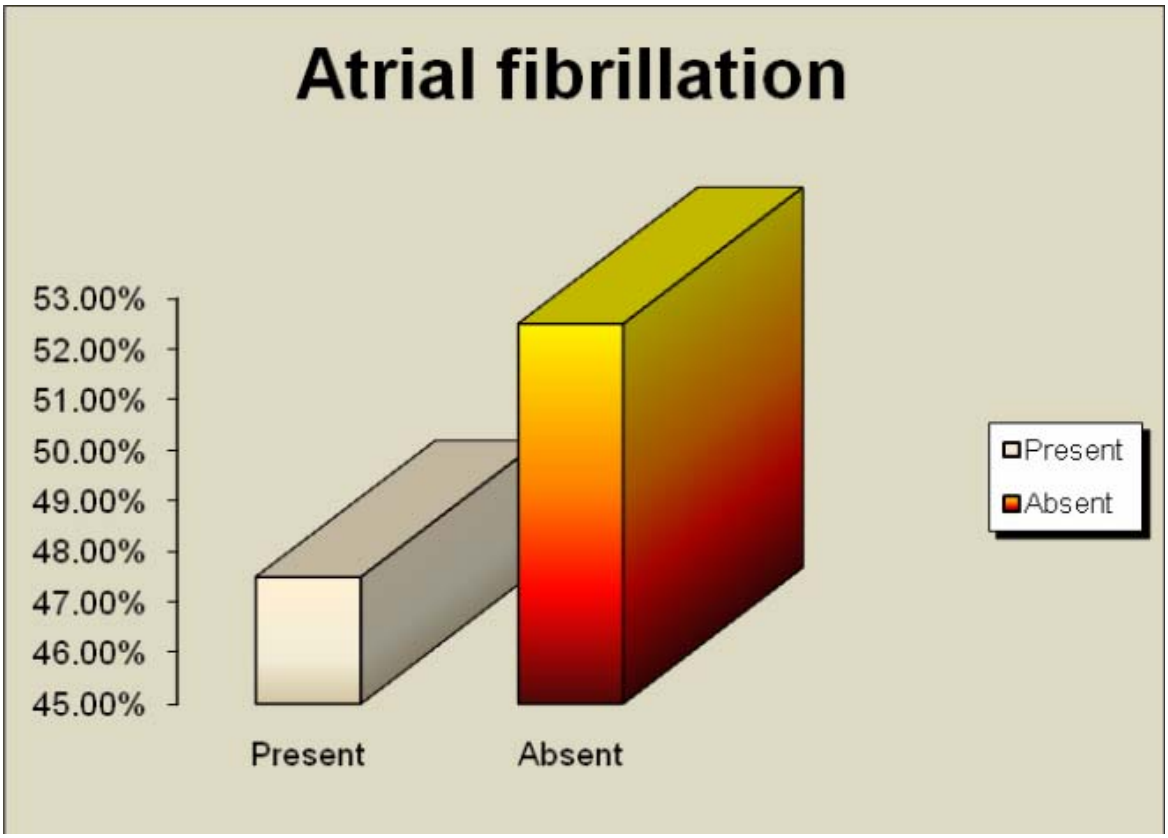



Age Distribution
27.50%
22.50%
50%
20-40
40-60
Above 60

Cardiac lesion	No of patients	Percentage
MS	9	22.5%
MR	1	2.5%
AS	0	0.0%
AR	0	0.0%
MS/MR	4	10.0%
AS/AR	3	7.5%
Multi valvular	9	22.5%
DCM	2	5.0%
MVP	1	2.5%
CAHD	11	27.5%



Atrial fibrillation	Number	Percentage
Present	19	47.5%
Absent	21	52.5%
Total	40	100%

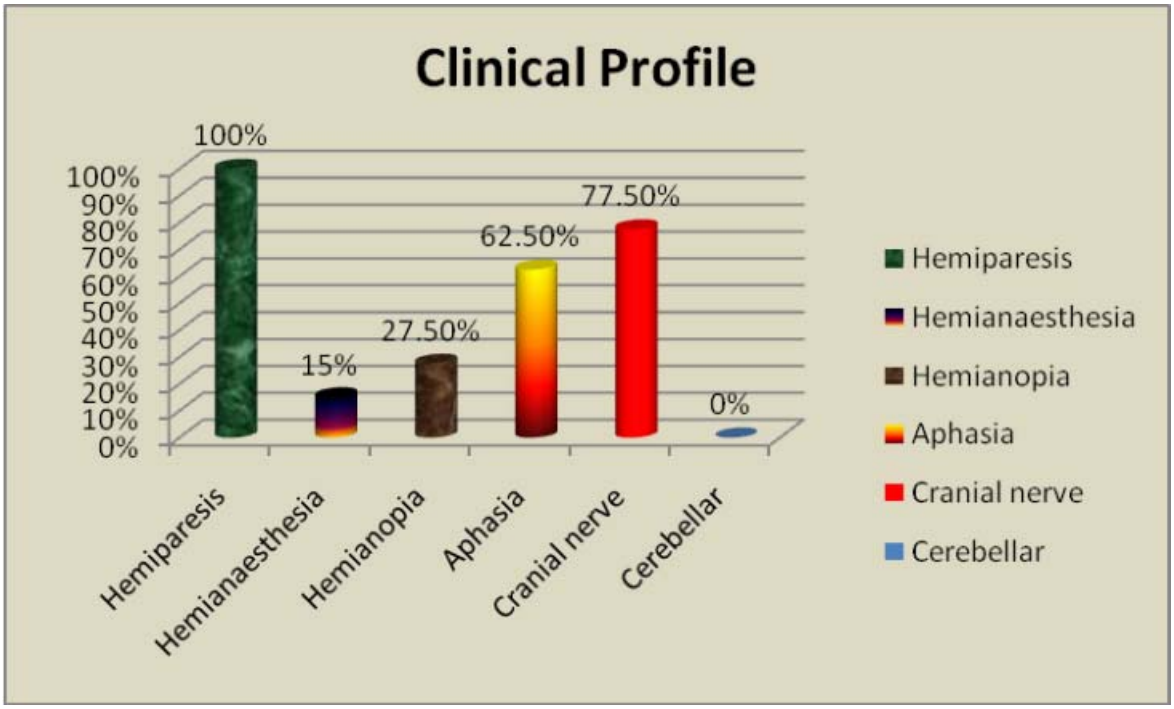


Recovery pattern

Recovery	**Number**	%
Dead	8	20
Static	12	30
Improved	20	50
Total	40	

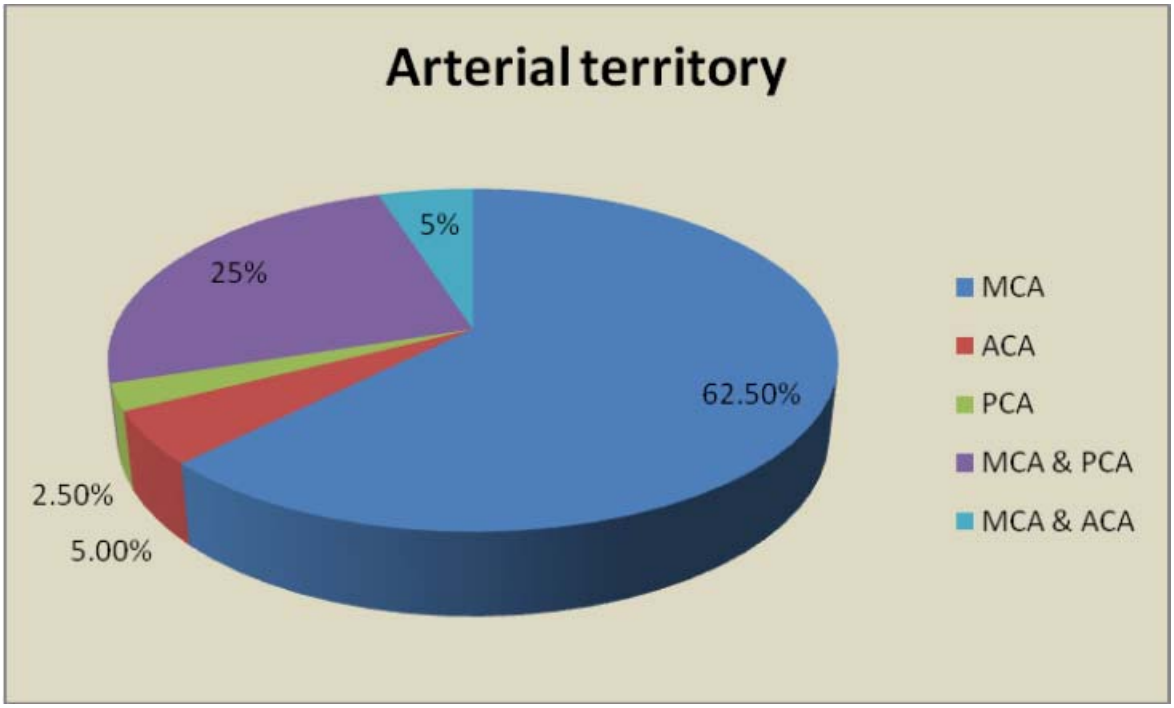
Clinical profile :

Hemiparesis	- 40(100%)
Hemianaesthesia	- 6(15%)
Hemianopia	- 11(27.5%)
Aphasia	- 25(62.5%)
Cranial nerve palsies	- 31(77.5%)
Cerebellar signs	- nil



Arterial territory :

MCA –	- 25(62.5%)
MCA&ACA	-2 (5%)
MCA & PCA	- 10(25%)
ACA	- 2(10%)
PCA	- 1(2.5%)



DISCUSSION

The present work is a longitudinal hospital based study focusing on pattern of evolution of cardio embolic stroke, clinical profile and cardiac lesions that caused the stroke.

These criteria for inclusion and exclusion were changes based on similar studies done from other centers in the world. Follow up was achieved after 4 weeks. Activities of daily living reported after 4 wks have been shown to correlate highly with those measured from direct examination. This novel method ensured a good follow up. Significant changes in the disability pattern were not expected during the immediate past stroke period. Hence 4wks was chosen as follow up period. By 6months the long term disability pattern was the same as that of 4wks.

In our prospective study of cardio embolic stroke, incidence of stroke was high among males (90%), especially above 45yrs (60%).

36 males and 4 females were included in this study. In males earliest age atwhich stroke appeared was 25 and highest age was 78.

Regarding age distribution, 20 patients were in the age group of 20 – 40 which was 50% next common age group to be involved in above 60 years having incidence of 27.5%.

Median age of presentation in males was 51. In females 37 was the youngest age to be affected by stroke and 66 was the eldest age at which stroke appeared. Median age of presentation in females was 50.

A study on stroke profile by department of internal medicine, Santiago, Chile shows that Out of 91 embolic stroke male – female ratio was 85;15% whereas in our study ratio is 90;10%.[7, 10, 28]

Regarding time of onset of stroke, most of the patients (15)gave history of appearance of symptoms in the morning hours i.e., 6am-2pm. Onset of stroke in the evening hours was also of same frequency in the time 2pm-10pm (15 patients.

Considering the importance of activity that led to the onset of stroke, most of the patients were doing their work(50%). 11of the patients gave history of rest at the onset of stroke(27.5%) 9 patients

were sleeping at the onset (22.5%) whereas studies show that embolic stroke is precipitated by height of emotion and work.[9,19]

In the analysis of clinical profile all the 40 patients were having hemiparesis or hemiplegia 6 patients were found to have sensory dysfunction. Speech disorder in the form of aphasia was present in 25 patients(62.5%). Only cranial nerve found to involved was UMN type of VII cranial nerve (77.5%). Cerebellar dysfunction was present in none of the patients. Visual disturbance in the form of homonymous hemianopia in 11 out of 40 cases.

In a study of clinical profile of stroke in eastern Nepal out of 150 cases hemiplegia was present in 49.3%. isolated cranial nerve palsy was present in 97 cases. The commenest nerve involved was facial nerve which was similar to finding to our study.[11, 29]

In our study considering etiology, Rheumatic valvular heart disease was the leading first (65%) among all other cardiac causes.

Ischemic heart disease (27.5%) was the second leading cause of cardio embolic stroke. [10,11,22]

Of the all the valvular heart disease multivalvular heart disease was the leading cause(25%). Among the combined causes mitral heart disease (10%) was the leading pathology followed by aortic (5%) (18)

In the isolated vaIvular pathology, mitral stenosis was the most common cause followed by mitral regurgidation which was proved in other studies also.(18,33)

Cardiomyopathy and MVP are the other causes with frequency of 5% and 2.5%. Whereas studies show that cardiomyopathy leads to stroke in > 10% of cardiac lesions. Cardiomyopathy was found to be causative in 2 of all cases. MVP was found in only one case. In a study of journal of ACC, cardiomyopathy led to stroke in significant proportion 12%. (12)

Coronary artery heart disease, in the form of ischemia and infarction, was predisposing to embolic stroke in 11 out of 40 cases.

In the study of stroke profile in chile valvular heart disease was present in 24cases. Atrial fibrillation was present in 67 cases. This study concluded that valvular heart diseases and atrial fibrillation were significant independent predictors of embolic stroke. Other

studies also concluded that rheumatic heart disease was important cause of embolic stroke .Among non rheumatic causes coronary heart disease commonly cause embolic stroke. In our study Of the 40 cases Atrial fibrillation was present in 19 cases(47.5%) which implies significance in aetiology of stroke as with other studies[6,10,12]. Among the Rheumatic heart disease, mitral valvular disease MS, MR or combined lesion may lead to stroke commonly is comparison with aortic diseases. [6,20]

Regarding the outcome 20 cases improved their power which was assessed with Barthell score (50%). 12cases were found to be static who were discharged with residual palsy (30%). 8cases died during the period of hospital stay(20%). Remaining 20 cases were found to be static in neurological examination. [20]

On considering arterial territory, most common arterial territory found to be involved was that of Middle Cerebral Artery i.e in 25 out of 40 cases(62.5%)%). Isolated Posterior cerebral artery was involved in 1 cases (2.5%)Least common was that of Anterior cerebral artery involved in 2cases(5%). MCA was involved in combination with ACA in 2 cases. MCA was involved in combination with PCA in

10 cases.Even though MCA territory was involved commonly in all cardio embolic stroke, 44% of PCA stroke was caused by cardio embolism[6].

A sudy in medical journal of Kathmandu university showed that out of 150 cases MCA territory was involved in 67 cases(77%). PCA was found to be involved in 10 cases(11.49%). ACA territory was found to be affected in 6 cases(6.8%). Combined involvement was found in 14 cases. MCA & ACA was the common combination. Our study proved MCA is the commonent territory involved in embolic stroke which was same finding given by study in Journal of Kathmandu University. [11,23]

In all the 40 cases clinical findings correlated with CT findings.

Specific cardiac lesions were not found to produce any specific clinical types of neurological illness. All the cardiac lesions produced similar neurological involvement in all cases.

CONCLUSION

1) Cardioembolic stroke is common among males and the median age of presentation is 51.earliest age reported was 25.
2) Onset of stroke in most of the cases(30patients) occurred during daytime hours 6am-10pm
3) Symptoms of stroke appeared during routine work in most cases(50%).
4) Hemiplegia was the commenest presentation and clinical profile correlated with CT findings in all cases. Among the cranialnerves facial nerve was the only nerve involved.
5) In this study rheumatic heart disease was found to be causative in 65% cases
6) Among rheumatic, multivalvular involvement was found in most cases rather than isolated valve involvement
7) In the isolated valvular lesions Mitral stenosis was predominant lesion
8) Atrial fibrillation was found in significant proportion of cases(47.5%)

9) As with other study arterial territory commonly involved was Middle cerebral artery that was confirmed by CT scan.

10) CAHD was present in significant proportion of cases.

11) No specific clinical profile was found with reference to specific cardiac lesion.

REFERENCES

1. Harrison's principles of internal medicine.
2. Adams and victor's principles of neurology
3. Online newspaper of Prof.Yasser Metwally – cardioembolic stroke
4. Journal of society of interventional radiology – study of stroke patients by MRI & echocardiography
5. Journal of API dec2007- study of PCA&ACA strokes
6. Essentials of clinical neurology by Weiber
7. Journal of BMC neurology- Retrospective study of stroke registries
8. Journal of BMC neurology – Clinical study of stroke patients ACA stroke
9. Journal of Arch neurol 1999 vol 56 no;7 – New England medical center registry of postr. Circulation stroke
10. Study on cardiovascular profile of occlusive vascular disorder by Corbalan R, Tapia J, Dept of Internal medicine,Santiago, Chile
11. Clinical profile of stroke in eastern Nepal; kathmandu university medical journal 2006

12. Clinical profile of stroke in 900 patients with cardiomyopathy ; journal of American college of cardiology vol 39 16-1-02 .
13. Mas JL et al; treatment cerebrovasular events associated with PFO, atrial septal aneurysm- newz neur journal 345, 2001.
14. Kelly, Roger etal, cardio embolic stroke; an update -southern medical journal Apr.2003 Vol 96 is 4
15. Aatif Husain et al, Transesophageal echo in cardio embolic stroke – Dept. of Neurology, Pensylvaria, USA,
16. J.P.Mohr, Dennis et al – stroke; pathophysiology diagnosis and management
17. cardio embolic stroke; an update regarding potential sources of cerebral embolism.
18. Megan C.Leasy et al; cardio embolic stroke an update an etiology ,diagnosis and management. annals of Indian academy of neurology2008;11-5
19. A Aroboix, I Paebilla etal; livecelll shows of 222patients with pure motor stroke.
20. Gran A.J, Buggle for etal; Risk factor, outcome and treatment in subtypes of ischemic stroke- German stroke data bank .
21. Brust JCM etal; ACA disease,
22. Lee B, Harvy S, et al – The Hallyton Stroke registry – Analysis of 1, 654 of cardic stroke.

23. Garcia Erolat et al; predicting a early neurological recovery after ischemic stroke
24. Bongonssalavasky J et al; ACA territory a infarction in laussame stroke registry; clinical & etiology pattern.
25. Kumaral E, Bay ulkam G eta;; spectrum of anterior cerebral artery infarction clinical & MRI findings.
26. Vemmos KN, Takis CT et al; The Athens stroke registry ; remoth of a five yr hospital based study
27. BMC neurol 2005; 5:9. cerebral infarction in diabetes; clinical pattern stroke sub types in hospital mortality.
28. Uma sundar, R. Mehatre etal; JAPI Etiopathogenesis and predictors of In hospital morbidity & mortality in PCA stroke.
29. Johann willeit, Ulmar etal; Neurological outcome of septic cardio embolic stroke Dept. of Neurology, Austria.
30. Ruttman, willeit et al; Neurological outcome of septic cardio embolic stroke after Infective endocarditis
31. Pasquale et al; cardiac causes of stroke -Division of cardiology, Bologn,Italyz.
32. Benjamin E.J etal; Mitral annular calcification and the risk of stroke New Eng. J. 1992; 32:37y
33. Kaul S et al Department of neurology, University of Maryland school of medicine, Baltimore, USA- clinical features of stroke

34. Nu Weir et al; Poat Graduate medical journal ; Foothills hospital, Canada

PROFORMA OF DATA

Name: **Age :** **Sex :**

Occupation:

Symptoms : **1)**

2)

3)

Time of onset:

Activity at onset:

Onset-admission interval:

Examination (CNS):

Higher functions:

Consciousness - EMV score -

Memory - immediate

Recent

Remote

Speech - Normal / Aphasic /Dysarthria

Orientation - Time / Space /Person

Cranial nerves -

Motor system - Rt Lt

Bulk

Arm

Fore arm

Thigh

Calf

Rt Lt

Tone :

UL

LL

Power :

UL

LL

Superficial reflexes :

Abdominal

Plantar

Cremasteric

DTR :

Biceps

Triceps

Supinator

Knee jerk

Ankle jerk

CVS examination;

Pulse;

BP;

Carotids;

Auscultation;

Investigations :

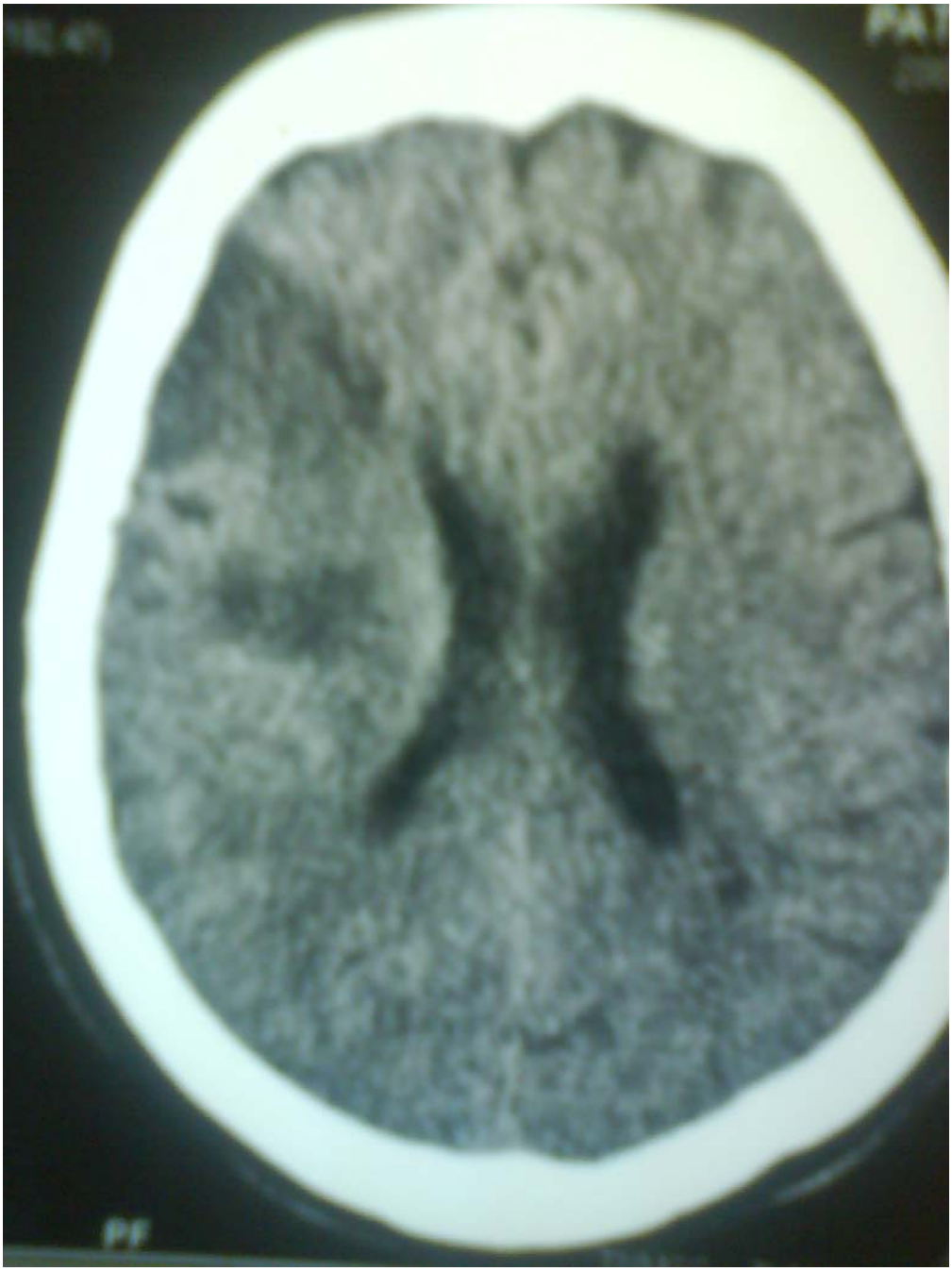
ECG :

X-ray chest :

Echo :

CT Brain :

Anterior cerebral artery



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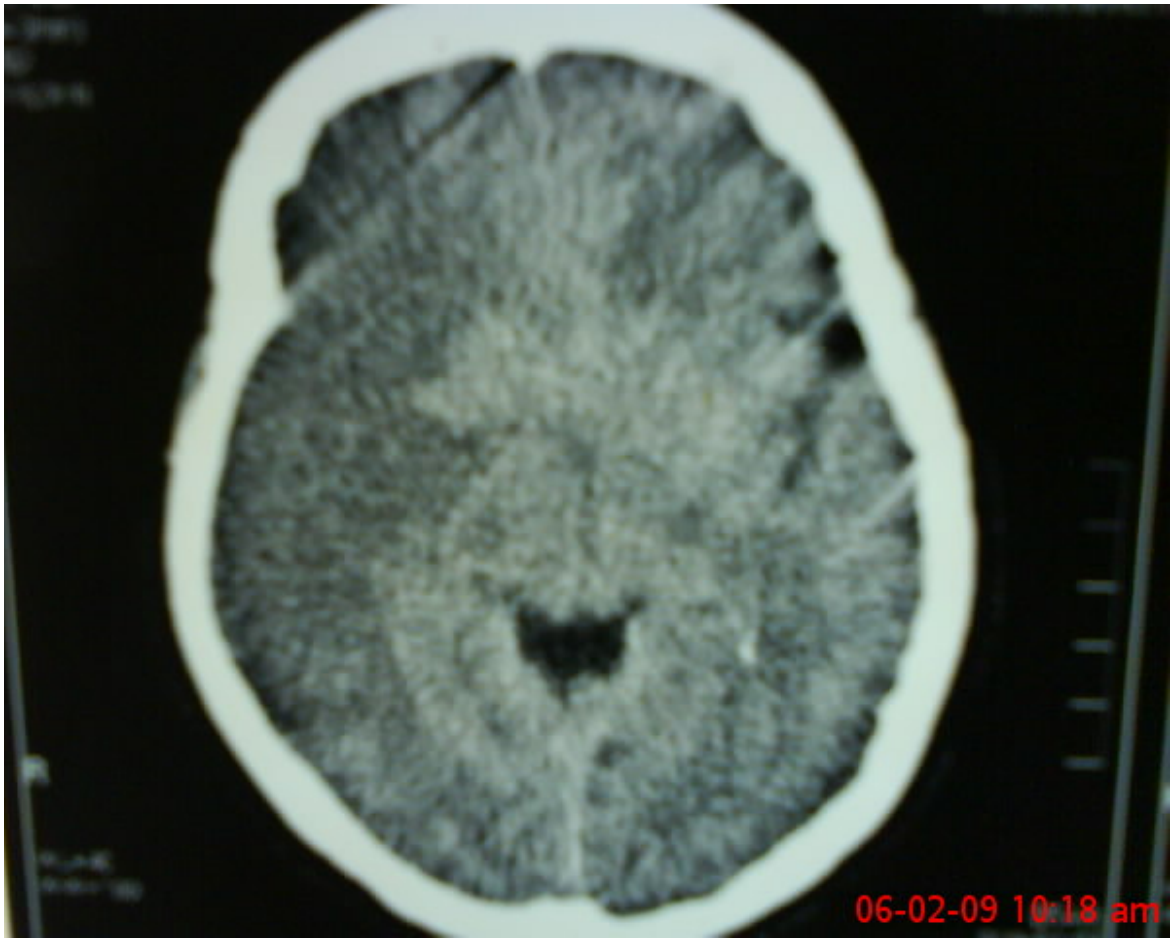
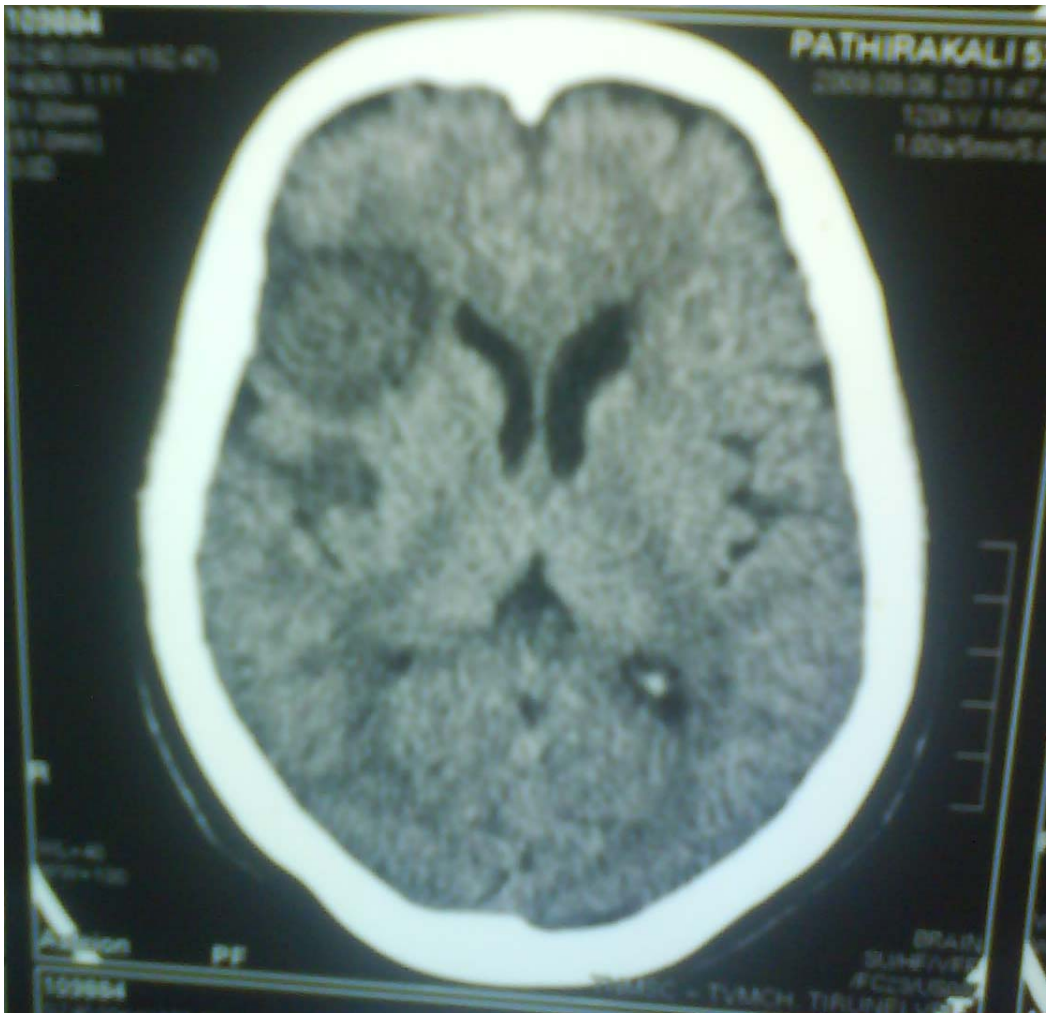
Middle cerebral artery and Anterior cerebral artery



Right middle cerebral artery

Left middle cerebral artery

Left posterior cerebral artery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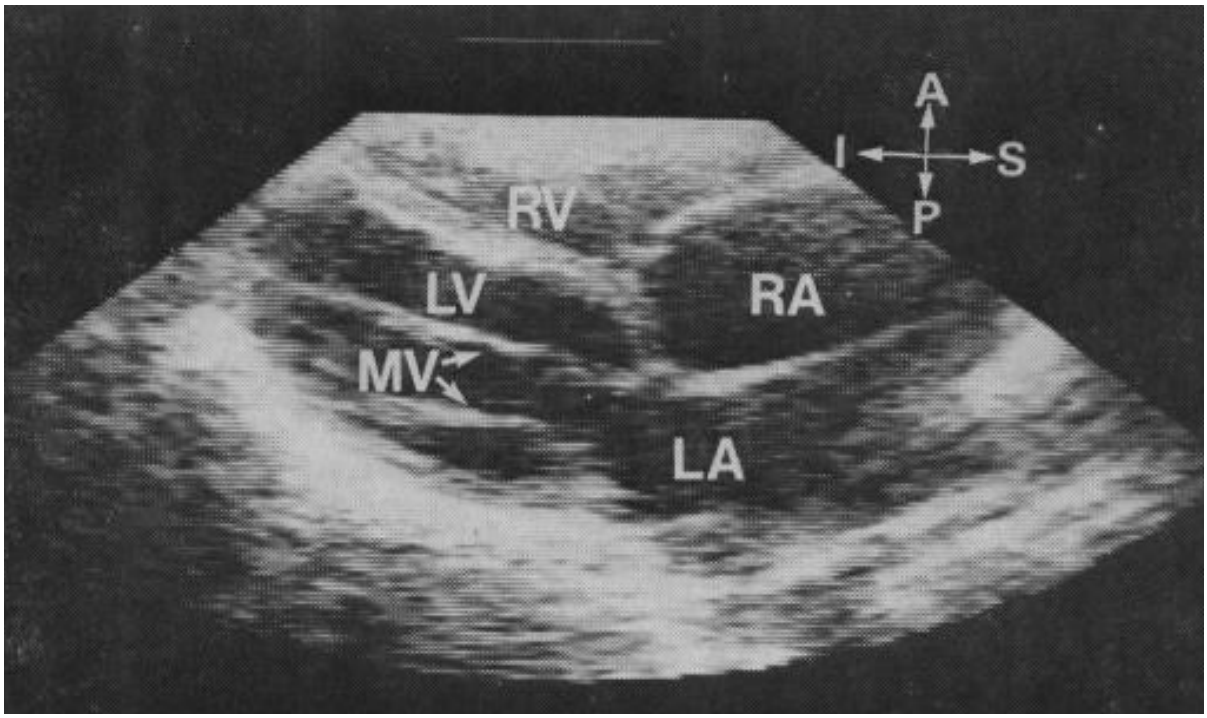
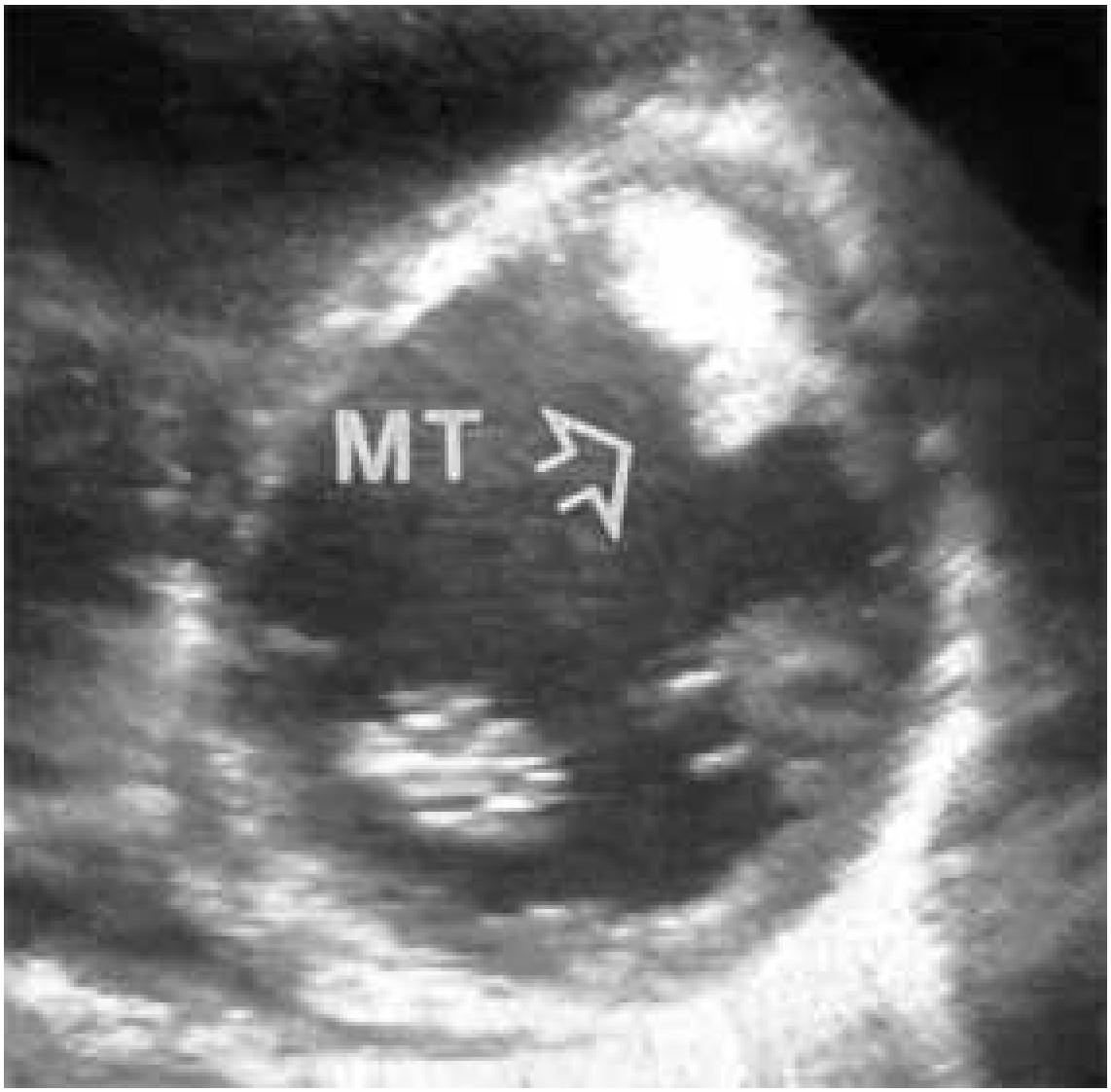
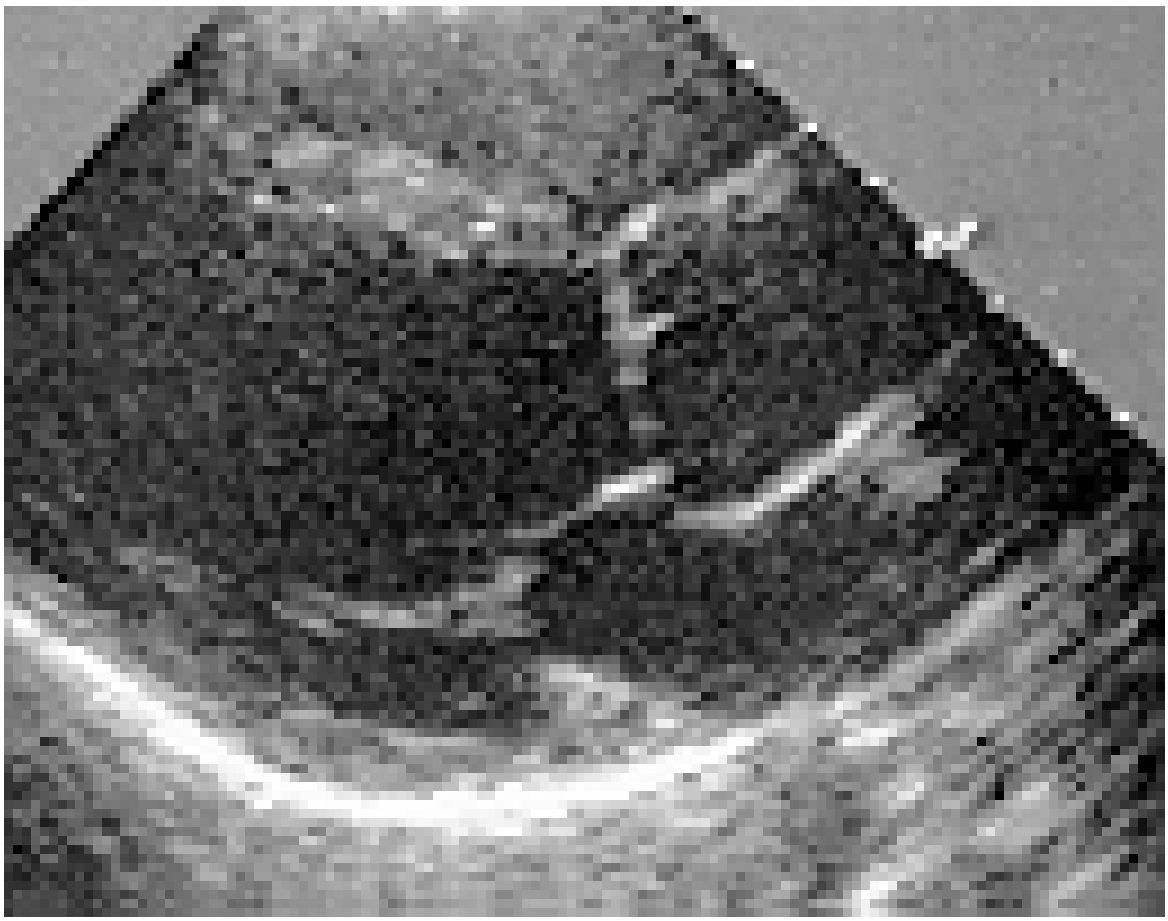
Mitral stenosis



LV Clot



Dilated cardiomyopathy



MASTER CHART

S. No	Name	Age	Sex	I.P.No.	D.O.A	Clinical profile	CT	Echo Cardiogram
1.	Mohammed kutti	75	M	282	09-01-08	Aphasia R UMN VII palsy R hemiplegia R hemianopia Hemianaesthesia	L MCA, PCA infarct	MS/MR/AR
2.	Muthu naicker	52	M	939	23-01-08	Aphasia R UMN VII palsy R hemiparesis	L MCA infarct/	MS/MR/AR
3.	Raja Mohamed	35	M	2037	23-01-08	R hemiparesis R UMN VII	L MCA	MS/MR/AR/PHT
4.	Selvaraj	50	M	4068	30-01-08	R UMN VII palsy R hemiparesis	L MCA	MS
5.	Mariappan	34	M	7239	06-02-08	L hemiparesis	R MCA	MS/PHT
6.	Jebakumar	39	M	7928	15-02-08	Aphasia R hemiparesis	L MCA	MS/MR
7.	Arumugam	60	M	8102	22-02-08	L hemiparesis	R MCA	MS/PHT
8.	Pathrakali	40	F 8	8139	26-02-08	L hemiparesis	ACA	MS/AS/AR
9.	Raviraj	45	M	10817	11-03-08	L UMN palsy LHemiparesis	R MCA	MS/MR/ LA clot
10	Sivanatchiammal	47	F	10999	26-03-08	Aphasia R hemiplegia UMN VII palsy Hemianaesthesia	L MCA	Dyskinesia antr wall
11	Ganesan	31	M	11002	27-03-08	L hemiparesis	R MCA	MVP
12	Yousuf sahib	85	M	11231	2-4-08	Aphasia R UMN VII palsy	L MCA	Dyskinesia antr wall

						R hemiplegia		
13	Annadurai	40	M	11312	09-04-08	R UMN palsy R hemiplegia R Hemianaesthesia	L MCA	MS/PHT
14	Muthuraman	48	M	11409	16-04-08	LUMN VII L hemiparesis	R MCA	MS/PHT
15	Jeyaraman	50	M	12429	07-05-08	Aphasia R UMN VII R hemiparesis hemianaesthesia	L MCA / ACA	AS/AR
16	Masthan	70	M	12499	14-05-08	Aphasia L umn VII L hemiplegia	R MCA R PCA	Antr wall dyskinesia
17	Subbiah	60	M	14572	28-05-08	Dysarthria R hemiparesis	L MCA	Antr wall dyskinesia
18	Muthiah chettiar	60	M	15242	-8-06-08	R UMN VII R hemiparesis Aphasia R Hemianopia	L MCA/ PCA	AS/AR
19	Raja ali	75	M	26455	17-06-08	R UMN VII R hemiparesis Aphasia	L MCA	MR/ AR LA clot
20	Chidambaram	67	M	19217	18-06-08	Aphasia R hemiplegia	L MCA	Antr wall dyskinesia
21	Veyil muthu	80	M	21312	25-06-08	Aphasia L umn VII L hemiplegia L Hemianopia	R MCA PCA	LA clot Antr wall dyskinesia
22	Natarajan	45	M	23108	09-07-2008	Aphasia L umn VII L hemiparesis	R MCA ACA	Aneurysm LV/ clot in LV/ AORTIC sclrosis

						L hemianaesthesia		
23	Kadher meeran	56	M	23489	23-07-08	Unconscious	L cerebellar	Dyskinesia antr wall
24	Suresh	21	M	23597	25-07-08	Aphasia L UMN VII L hemiplegia Lhemianopia	R MCA/PCA	DCM/MR
25	Shenbagam	66	F	45954	30-07-08	semiconscious	R MCA/parietal infarct	MS/TR/PHT
26	Muthukrishnan	58	M	33626	05.08.08	Aphasia L UMN VII L hemiplegia L hemianaesthesia	R MCA/Corona radiate	MS/PHT
27	Balasubramanian	48	M	21955	21.08.08	R hemiparesis R UMN VII	L ACA	MS/PHT
28	Thangapandi	48	M	33682	06.08.08	L UMN VII L hemiplegia	R MCA	MR
29	Chellakutti Devar	65	M	34640	13.08.08	L UMN VII L hemiplegia hemianopia	R MCA / PCA	MS/MR/AR/PHT
30	Sundaram	35	M	34602	20.08.08	Aphasia R UMN VII R hemiparasis R hemianopia	L MCA / PCA	MS/MR/PHT
31	Subbiah	55	M	34765	26.08.09	Aphasia R UMN VII R hemiplegia	L MCA	Anterior wall Dyskinesia
32	Abdul Kalam	23	M	36615	26.08.08	R hemiparasis R UMN VII	L MCA	AS/AR
33	Natarajan	65	M	46624	03.11.08	Aphasia R UMN VII	L MCA / PCA	Anterior wall Dyskinesia

						R hemiplegia R hemianopia		
34	Antony Yagappar	43	M	47911	11.11.08	Aphasia R UMN VII R hemiplegia	L MCA / Corona radiata	MS/AR/MR/PHT
35	Sappani	50	M	13989	08.04.09	R UMN VII R hemiparasis	L MCA	Anterior wall Dyskinesia
36	Sargunam	38	M	18315	18.05.09	L UMN VII L hemianopia L hemiplegia	R MCA/PCA	MS/dilated LA
37	Shunmugaiah	65	M	23290	10.06.09	Aphasia R UMN VII R hemiplegia	L MCA	MR/AR/CAHD/LV disfunction
38	Marimuthu	60	M	23489	15.06.09	Aphasia L hemiplegia L UMN VII	R MCA	Anterior wall Dyskinesia
39	Vetrivel	37	F	36386	06.09.09	Dysarthria L UMN VII R hemiparasis L hemiplegia hemianopia	R PCA / MCA	MS/MR/AR/TR/PHT
40	Lakhumanan	45	M	37539	13.09.09	R hemiparasis R UMN VII	L MCA/	MVP